Lecture 1 *Pathology of Thrombosis*

Topic	Key points (from your text)	High-yield anchors
Big picture	**Blood coagulation is physiologic** and essential for hemostasis and survival. It becomes **pathologic** when **unnecessary clotting** is activated → injury/harm.	**Physiologic vs pathologic thrombosis**
Core pathogenesis	**Virchow's triad** = 3 interacting causes of pathological thrombosis. A patient may show **one, two, or all three**. (Mnemonic given: **SHE**).	**Stasis/turbulence** + **Hypercoagulability** + **Endothelial injury**
Endothelium in normal state	Under **basal** conditions, endothelial cells create a **non-adhesive, non-thrombogenic** surface → protects from unnecessary thrombosis. Maintained by stable microenvironment: **normal BP**, **normal laminar flow**, and **growth factors** supporting endothelial integrity.	**Normal endothelium is anti-thrombotic**
Endothelium in pathological state	**Endothelial injury → endothelial activation.** Once activated, endothelium promotes thrombosis via increased expression of: **pro-coagulant factors**, **adhesion molecules**, **pro-inflammatory factors**, and **chemokines/cytokines/growth factors**.	**Injury → activation → pro-thrombotic shift**
Vascular wall response to injury	Injury affects **endothelium + smooth muscle**. Smooth muscle response: **migrate** from media → intima, **proliferate** in intima, **produce ECM proteins**. This thickens intima → **narrows lumen** → reduced flow.	**Intimal thickening → luminal stenosis**
Consequences of excessive healing	Healing can be pathologic: **excessive intimal thickening → stenosis/occlusion** → ↓ flow.	**Ischemia → necrosis → possible infarction**
Causes of endothelial injury	Listed causes: **valvulitis**, **MI**, **atherosclerosis**, **traumatic/inflammatory conditions**, **hypertension**, **endotoxins**, **hypercholesterolemia**, **radiation**, **smoking**, etc.	**Atherosclerosis, HTN, smoking, MI** (classic exam drivers)
Normal blood flow	**Laminar flow** = parallel layers with different velocities: **fastest in center**, **slowest near wall**. Keeps **platelets central** and away from endothelium → **reduces thrombosis risk**.	**Laminar flow is protective**
Turbulence: narrowed vessel	Thickened wall **narrows lumen** → **chaotic** direction/velocity → higher	**Atherosclerotic narrowing →**

	thrombosis risk. **Atherosclerosis** is classic example.	**turbulence → thrombosis**
Turbulence/disturbance: dilated vessel	**Aneurysm** → dilated wall → **irregular + sluggish** flow (disturbed flow) → promotes thrombosis.	**Aneurysm-associated disturbed flow**
Stasis definition & effects	**Stasis** = slower-than-normal flow. Major factor for **venous thrombi**. In stasis: **reduced delivery of anti-clotting factors**, **thrombi become stronger/more stable**, **fibrinolysis weaker**.	**Stasis = venous thrombosis driver**
Causes of stasis	Listed: **atherosclerosis**, **aneurysms**, **MI** (non-contractile fibers), **mitral stenosis** (atrial dilation), **hyperviscosity** (↑ PCV) and **sickle cell anemia**, etc.	**MI, mitral stenosis, hyperviscosity**
Hypercoagulability definition	**Increased tendency to form clots** vs normal population. Can be **inherited or acquired**.	**Primary vs secondary hypercoagulability**
Primary (genetic) hypercoagulability	**Inherited mutations** in clotting/anti-clotting factors. **Factor V gene** and **prothrombin gene mutations** are **most common primary causes**.	**Factor V** + **Prothrombin** (very high yield)
Secondary (acquired) hypercoagulability	**More frequent** than primary. Often **multifactorial**. Causes listed: **immobilization**, **MI**, **AF/arrhythmia**, **surgery**, **fractures**, **burns**, **cancer**, **prosthetic cardiac valves**, etc.	**Cancer, immobilization, surgery, AF**
Where thrombi form	Can develop anywhere in CVS: **cardiac chambers**, **valves**, **arteries**, **veins**, **capillaries**.	**Location predicts trigger**
Arterial/cardiac thrombi (trigger)	Begin at sites of **endothelial injury or turbulence**, usually **superimposed on an atherosclerotic plaque**.	**Endothelial injury + atherosclerosis**
Venous thrombi (trigger)	Occur at sites of **stasis**. Most commonly **lower extremity veins (90%)**.	**Lower extremities ~90%**
Attachment & embolic risk	Thrombi are **focally attached** to underlying surface. The **propagating portion is poorly attached** → prone to **fragmentation → embolus**.	**Propagation tail = embolic danger**
Lines of Zahn	**Gross/microscopic laminations**: **alternating pale platelet/fibrin layers** with **darker RBC-rich layers**.	**Distinguish antemortem thrombus from postmortem clot**
Postmortem clots	**Non-laminated** (no Lines of Zahn).	**No Lines of Zahn = postmortem**

Mural thrombi	**In heart chambers or aortic lumen**. Definition: thrombus attached to wall of chamber/vessel.	**"Mural" = wall-adherent**
Cardiac vegetations	**Thrombi on heart valves**. Composition: **fibrin + platelets + inflammatory cells ± microorganisms**.	**Valve thrombi = vegetations**
Types of vegetations	1) **Infectious** (bacterial/fungal blood-borne) e.g., infective endocarditis → **large, friable, destructive**. 2) **Non-infectious** e.g., **rheumatic**; **NBTE** → **small, bland, non-destructive**.	**Infectious = big/destructive** vs **NBTE = small/bland**
Fates of thrombi	1) **Propagation** → more platelets/fibrin → obstruction. 2) **Embolization** → dislodge/fragment. 3) **Dissolution** → fibrinolysis (**only in recent thrombi**). 4) **Organization & recanalization** → inflammation/fibrosis; flow may partially restore or thrombus incorporated into thickened wall. 5) **Superimposed infection → mycotic aneurysm**.	**5 classic outcomes**
Organization (definition)	Ingrowth of **collagen**, **ECM proteins**, **new endothelial cells**, **smooth cells**, **fibroblasts** into **fibrin-rich thrombus** → permanent structural change.	**Organization = permanent remodeling**
Mycotic aneurysm	**Infectious aneurysm**. "Mycotic" = **infection-related (not necessarily fungal)**. "Aneurysm" = **abnormal dilation**.	**Infection + vessel wall weakening**

Lecture 2 *Embolism & Infarction*

Topic	Key points (from your text)	High-yield anchors
Embolus definition	**An embolus** is a **detached intravascular solid, liquid, or gaseous** mass carried by blood to a site **distant** from origin.	**Detached + travels**
Emboli types (by composition)	1) **Thromboembolism: 99%** (dislodged thrombus) 2) **Fat embolism** 3) **Air/Nitrogen embolism** 4) **Amniotic fluid embolism**.	**99% = thromboembolism**
Quick mnemonic in text	Embolus moves like a **FAT BAT**. Causes list: **Fat	Air
Emboli types (by origin site)	**Venous** vs **Arterial (systemic)**. Two circulation sides.	**Venous vs arterial classification**
Venous emboli: origin & target	**Most venous emboli originate from lower limbs** (esp. DVT). **Target = lungs**.	**Lower limb DVT → lungs**

Arterial emboli: origin & target	**Most arterial emboli originate from heart chambers**. **Target most often lower limbs (75%)**.	**Heart → systemic organs**
General effect on blood flow	Emboli cause **partial or complete vascular occlusion → ischemic necrosis (infarction)** of downstream tissue.	**Occlusion → infarction**
Pulmonary thromboembolism origin	**95%** originate from **deep vein thrombi of lower limbs (DVT)**.	**DVT is the central source**
Saddle embolus	**Large embolus at bifurcation of pulmonary artery trunk → fatal**.	**Saddle = catastrophic PE**
Paradoxical embolus	**Venous → systemic** through **PFO (Patent foramen ovale), ASD (Atrial septal defect), VSD (Ventricular septal defect)**. "Paradoxical" because venous clot **bypasses lungs** via shunt → systemic arterial blockage (e.g., brain).	**Right-to-left shunt concept**
PE clinical outcomes (range)	**Asymptomatic 60–80%** (small). Can cause **pulmonary infarction** (large), **pulmonary hemorrhage**, **pulmonary hypertension + RV failure** (showers over long time), **sudden death** when **>60% of pulmonary vessels obstructed**.	**Size + burden + chronicity matter**
Determinants of thromboembolism outcome	Depends on: **size**, **site of occlusion**, **number of emboli**, **acute vs chronic/recurrent**.	**4-variable exam framework**
Symptoms linked to DVT/PE in text	**Loss of consciousness** may occur due to **hypoxia** when critical flow blocked. **Calf/thigh pain** in DVT from venous blockage + inflammation.	**Clinical clue integration**
AF → arterial emboli mechanism	**Atrial fibrillation** → abnormal atrial contraction + dilation → **stasis in left atrium → thrombus** → may embolize **LA → LV → systemic** → lodge in **brain, kidneys, small intestine** etc.	**AF = classic cardioembolic source**
Systemic (arterial) thromboembolism	**80% due to intracardiac mural thrombi**. Causes listed: **2/3 Lt ventricular failure**, **¼ Lt atrial dilatation**, **ulcerated atherosclerotic plaque**, **aortic aneurysm**, **valve vegetation** etc.	**Mural thrombi dominate**
Major arterial targets	**Lower limbs, brain, intestine, kidneys, spleen** — essentially **any organ with arterial supply**.	**Lower limb + brain high yield**
Fat embolism definition	**Fat globules** in **lung or systemic** circulation.	**Fat in vessels**
Fat embolism causes	**Long bone fractures**; less commonly **adipose injury** (e.g., **fat necrosis in acute pancreatitis**).	**Long bones = key trigger**
Fat embolism mechanisms	1) **Mechanical obstruction** 2) **Free fatty acid release → toxic endothelial injury + strong systemic immune response**.	**Mechanical + biochemical injury**

Frequency vs syndrome	After skeletal injury, fat embolism occurs in **~90%**, but **clinical fat embolism syndrome (FES) <10%**.	**Common finding; rare syndrome**
FES composition clue	**FAT EMBOLUS = fat globules + hematopoietic cells**.	**Histology cue**
FES clinical triad + others	**Pulmonary insufficiency**, **neurologic symptoms**, **petechial rash**; plus **fever, anemia, thrombocytopenia**. **Symptoms 1–3 days after injury**. **Death ~10%**.	**Classic triad + timing**
FES therapy	**No specific treatment**. **Prevention + early diagnosis + supportive care**. Support includes **oxygenation/ventilation**, **stable hemodynamics**, **blood products if needed**, **hydration**, **DVT + stress GI bleed prophylaxis**, **nutrition**.	**Supportive care is mainstay**
Air embolism core idea	**Mechanical vascular obstruction**.	**Air blocks flow**
Air embolism causes	**Surgical/obstetric procedures**, **traumatic chest wall injury**, **decompression sickness** (nitrogen) in deep-sea divers.	**Iatrogenic + diving**
Decompression sickness mechanism	High pressure dissolves nitrogen in blood; **slow ascent** allows nitrogen to leave via lungs; **rapid ascent** → nitrogen bubbles form → **nitrogen embolism**.	**Pathophys favorite**
Air embolism consequences	1) **Painful joints** (bubbles in muscles/supporting tissues) 2) **Focal ischemia brain/heart** 3) **Respiratory distress ("chokes")** with **lung edema/hemorrhage/atelectasis/emphysema** 4) **Caisson disease**: bone emboli → ischemic necrosis, often **femur, tibia, humerus heads**.	**"Bends" vs "chokes"**
Amniotic fluid embolism (AFE)	**Very rare**, during labor (e.g., C-section). **High mortality 20–40%**. Mechanism: **strong immune reaction + marked coagulation activation** + possible mechanical obstruction. Entry via **tears in placental membranes + rupture of uterine veins**.	**Obstetric emergency**
AFE clinical picture	**Sudden severe dyspnea**, **cyanosis**, **ARDS**, **hypotensive shock**, then **seizures, DIC, coma**.	**Respiratory collapse + DIC pattern**
AFE microscopy	**Fetal squamous cells**, **lanugo hair**, **fat**, **mucin** in maternal pulmonary microcirculation.	**Diagnostic autopsy clue**
Infarction definition	**Infarct** = **ischemic necrosis** from **arterial occlusion** or **venous drainage occlusion**.	**Arterial or venous blockage**
Infarction causes	**99%** from **thrombotic/embolic events**. Others: **vasospasm**, **atheroma expansion**, **extrinsic compression** (tumor), **vessel twisting** (**testicular torsion**, **bowel volvulus**), **traumatic rupture**.	**99% thrombo-embolic**

Infarct morphology overview	**Red (hemorrhagic)** vs **White (anemic)**; **Septic** vs **Bland**. Often **wedge-shaped**. **Margins become defined with time**.	**Color + infection status**
Histologic hallmark	**Ischemic coagulative necrosis** → eventually **scar**. **Brain exception: liquefactive necrosis**.	**Coagulative except brain**
Red infarcts: when occur	1) **Venous occlusions** (e.g., ovarian torsion) 2) **Loose tissues** (lung) 3) **Dual circulations** (lung, small intestine) 4) **Previously congested tissues** 5) **Reperfusion after arterial occlusion**.	**Lung + dual supply + venous block**
White infarcts: when occur	**Arterial occlusions in solid organs**: **heart, spleen, kidney**.	**Heart/spleen/kidney**
Septic infarcts	Occur when infarct is **superimposed by infection**: **infected vegetations** or **microbes seeding necrotic tissue** → infarct may become **abscess** with stronger inflammation.	**Infected emboli → abscess risk**
Outcome example	**Kidney white infarct** may be replaced by a **large fibrotic scar**.	**Scar formation in solid organs**
Factors influencing infarct development	1) **Nature of vascular supply** 2) **Rate of occlusion** (collaterals) 3) **Tissue vulnerability to hypoxia**: **neurons ~3 minutes**, **myocardium 20–30 minutes** 4) **Oxygen content of blood**.	**Time-to-irreversibility**
Classification trick Q&A in text	**Pulmonary artery embolus** is classified as **venous embolism** with **lungs as target**, because pulmonary artery carries **venous blood** from right heart to lungs.	**Pulmonary artery ≠ "arterial origin" assumption**
Can lung be a target of arterial emboli?	**Yes**: lung can be target of **venous** emboli (via right heart) or **arterial** emboli if coming from **left heart** reaching lung via **bronchial circulation**.	**Dual pathway concept**

Lecture 3 *Pathology of Veins & Lymphatics*

Topic	Key points (from your text)	High-yield anchors
Vessel wall basics	**Tunica media**: mainly **smooth muscle**, and in some vessels (especially **elastic arteries**) also **elastic fibers**.	**Media composition**
Artery vs vein (gross/functional)	**Arteries**: **thicker wall**, more **rounded/rigid**. **Veins**: **thinner wall**, **less-developed tunica media**, commonly **collapsed**.	**Artery thick/round vs vein thin/collapsed**
Normal vein physiology	**Venous valves** prevent backward flow and are **aided by surrounding muscles** to push blood and maintain function.	**Valves + muscle pump**

Varicose veins – definition	**Abnormally dilated, tortuous veins** due to **prolonged ↑ intraluminal pressure** and **loss of vessel wall support**.	**Pressure + wall support failure**
Varicose veins – most common site	**Superficial veins of the leg** are most typically involved.	**Superficial leg veins**
Varicose veins – symptoms/impact	**Venous stasis + edema** (simple orthostatic edema) and **cosmetic effect** (major complaint).	**Stasis edema + cosmetic**
Varicose veins – epidemiology	**10–20% of adult males** and **>30% of adult females** develop lower extremity varicosities.	**Very common, female predominance**
Varicose veins – risk factors	**Obesity**, **female gender**, **pregnancy**, **familial tendency**. **Premature varicosities** can result from **imperfect venous wall development**.	**Obesity + pregnancy + family history**
Varicose veins – microscopic morphology	**Vein wall thinning**, **intimal fibrosis** in adjacent segments, **spotty medial calcifications (phlebosclerosis)**, **focal intraluminal thrombosis**, **venous valve deformities** (rolling/shortening).	**Valve deformity + wall thinning + phlebosclerosis**
Varicose veins – complications	**Stasis, congestion, edema, pain, thrombosis**. **Chronic varicose ulcers** overlying varicosities in long-term patients. **Embolism is very rare**.	**Ulcers + thrombosis; embolism uncommon**
Thrombophlebitis & phlebothrombosis – terminology	**Interchangeable terms** in this lecture.	**Know both names**
Definition	**Inflammation + thrombosis of veins**.	**Inflammation + clot**
Most common site	**Deep leg veins (90% of all)**.	**DVT dominance**
Predispositions	**Congestive heart failure**, **neoplasia**, **pregnancy**, **obesity**, **postoperative state**, **prolonged bed rest/immobilization**.	**Immobility + cancer + surgery**
Local manifestations	**Distal edema**, **cyanosis**, **superficial vein dilation**, **heat**, **tenderness**, **redness**, **swelling**, **pain**.	**Classic inflammatory DVT signs**
Upper limb thrombophlebitis	Usually linked to **local factors** like **catheter/cannula**; sometimes related to **systemic hypercoagulability**.	**Think iatrogenic**
Special type 1: Migratory thrombophlebitis (Trousseau sign)	**Paraneoplastic hypercoagulability** due to tumor **procoagulant factors** (e.g., **colon**, **pancreatic cancer**). Leads to **multiple recurrent sites** of thrombophlebitis (extremities, abdomen, internal organs) with **intervals** between episodes.	**Cancer clue = Trousseau**
Special type 2: SVC syndrome	**Neoplasms compress/invade SVC**; **most common lung cancer**. Causes **marked**	**Lung cancer → SVC syndrome**

	dilation of head/neck/arm veins + cyanosis. **Key sign: Pemberton's sign** (facial congestion/distress upon arm elevation).	
Special type 3: IVC syndrome	**Neoplasms compress/invade IVC**; m/c highlighted: **hepatocellular carcinoma** and **renal cell carcinoma** (noted tendency to grow within veins). Findings: **marked lower limb edema** + **distention of superficial collateral veins of lower abdomen** ("**medusa**").	**RCC/HCC → IVC obstruction**
Lymphatic system – core function	Returns **excess interstitial fluid** to **venous circulation** → back to heart. **Obstruction** → fluid accumulation → **swelling + inflammation** below blockage → **lymphedema**.	**Obstruction → lymphedema**
Major lymphatic pathologies in lecture	1) **Lymphedema** 2) **Lymphangitis** 3) **Chylous** accumulations.	**3-item framework**
Lymphedema – primary	**Congenital** due to **lymphatic agenesis or hypoplasia**.	**Agenesis/hypoplasia**
Lymphedema – secondary (more common)	**Obstruction** of previously normal lymphatics. Examples: **malignant tumors**, **lymph node removal** (e.g., **mastectomy with ipsilateral nodes**), **post-irradiation**, **fibrosis**, **filariasis**, **post-inflammatory thrombosis/scarring**.	**Cancer + surgery + radiation + filariasis**
Lymphangitis – definition	**Acute inflammation** from **bacterial infections spreading into lymphatics**.	**Infection tracks lymphatics**
Most common organism	**Group A β-hemolytic streptococci**.	**GAS**
Morphology & clinical picture	Lymphatics **dilated** and filled with **neutrophils + monocytes**. **Red painful subcutaneous streaks** + **painful draining node enlargement (acute lymphadenitis)**. Can progress into venous circulation → **bacteremia or sepsis**.	**Red streaks + tender nodes**
Chylous accumulations – definition	**Milky lymph collections** in body cavities from **rupture of dilated lymphatics**, typically obstructed by **infiltrating tumor mass**.	**Tumor-associated lymph rupture**
Types	**Chylous ascites** (abdomen), **chylothorax** (chest), **chylopericardium** (pericardium).	**3 classic sites**

Lecture 4 arteriosclerosis

Topic	Key Concepts (Integrated From All Lectures)	High-Yield Anchors (Bold)
Normal Vessel Structure	Arteries have **thick tunica media** → more smooth muscle + elasticity for pressure regulation. Veins have **thin media**, collapse easily, rely on **valves + muscle pump**.	**Tunica media thickness**, **valves prevent backflow**
Thrombosis – Definition	Pathological blood clot formation due to abnormal activation of coagulation.	**Virchow's Triad (SHE)**
Virchow's Triad	1) **Stasis/Turbulence** 2) **Hypercoagulability** 3) **Endothelial Injury**. Each alone can cause thrombosis; together ↑↑ risk.	**Stasis**, **Hypercoagulability**, **Endothelial injury**
Endothelial Role	Normal endothelium = **anti-thrombotic**; injured endothelium = **pro-coagulant** (↑ adhesion molecules, cytokines, tissue factor).	**Injury → activation → thrombosis**
Arterial vs Venous Thrombi	Arterial thrombi → begin at **injury/turbulence**, superimposed on **atherosclerotic plaque**. Venous thrombi → due to **stasis**, especially **lower extremities (90%)**.	**Arterial = plaques**, **Venous = stasis, DVT**
Lines of Zahn	Alternating pale platelet/fibrin + dark RBC layers → **only in antemortem thrombi**.	**Distinguishes antemortem vs postmortem**
Fates of Thrombus	**Propagation**, **Embolization**, **Dissolution**, **Organization/Recanalization**, **Mycotic aneurysm**.	**5 classical outcomes**
Embolism – Definition	Detached **solid, liquid, gas** mass traveling in blood → occludes distant vessel.	**Thromboembolism = 99%**
Types of Emboli	**Thrombus (99%)**, **Fat**, **Air/Nitrogen**, **Amniotic fluid**.	**FAT BAT mnemonic**
Venous Emboli	Originate mostly from **DVT of lower limbs** → target **lungs** → pulmonary embolism.	**DVT → lungs**
Pulmonary Embolism	Small: **asymptomatic**; large: **pulmonary infarct, hemorrhage**; recurrent: **pulmonary HTN**; massive: **sudden death when >60% blocked**.	**>60% obstruction = fatal**
Saddle Embolus	Large embolus at **bifurcation of pulmonary artery → fatal**.	**Classic killer PE**
Paradoxical Embolus	Venous embolus crosses **PFO/ASD/VSD** → enters systemic circulation → stroke.	**Right-to-left shunt**
Arterial (Systemic) Emboli	**80% from intracardiac mural thrombi** (LV failure, LA dilation), targets: **lower limbs (75%)**, brain, kidneys, intestine, spleen.	**Heart → systemic organs**

Fat Embolism Syndrome	After long bone fractures: **respiratory distress, neurological signs, petechial rash** → appears **1–3 days later**.	**Triad: lungs + brain + rash**
Air/Nitrogen Embolism	Caused by surgery, trauma, rapid ascent (diving). Nitrogen bubbles → **"bends", "chokes", Caisson disease"**.	**Decompression sickness**
Amniotic Fluid Embolism	Rare but lethal (**20–40%**). Causes **DIC + ARDS + shock**. Histology: **fetal squamous cells, lanugo hair** in mother's lungs.	**Obstetric emergency**
Infarction – Definition	**Ischemic necrosis** due to occlusion of **arterial supply or venous drainage**. **99% from thromboembolism**.	**Coagulative necrosis (except brain)**
Red vs White Infarcts	**Red**: venous occlusion, loose tissues (lung), dual supply, reperfusion. **White**: solid organs (heart, spleen, kidney).	**Red = lung**, **White = heart/kidney**
Veins – Varicose Veins	Dilated, tortuous superficial veins due to ↑ **intraluminal pressure + wall weakness**. Risk factors: **obesity, pregnancy, female sex, genetics**.	**Lower limb superficial veins**
Varicose Complications	**Stasis, edema, pain, thrombosis, chronic ulcers**. **Embolism rare**.	**Chronic venous ulcers**
Thrombophlebitis / Phlebothrombosis	Inflammation + thrombosis of veins. **90% in deep leg veins**. Risk: **surgery, cancer, pregnancy, immobility**.	**DVT inflammation**
Migratory Thrombophlebitis (Trousseau)	Paraneoplastic **hypercoagulability**, especially **pancreatic & colon cancers**.	**Trousseau = visceral cancer**
SVC Syndrome	Compression by thoracic tumors (esp. **lung cancer**) → **head/neck vein distention**, cyanosis.	**Pemberton sign**
IVC Syndrome	Compression by **HCC or RCC → leg edema + abdominal vein distention ("medusa")**.	**RCC loves invading veins**
Lymphedema	Primary (congenital) or secondary (**tumors, mastectomy, radiation, filariasis**).	**Post-mastectomy lymphedema**
Lymphangitis	Infection of lymphatics by **Group A β-hemolytic streptococci → red streaks + painful nodes** → may cause **bacteremia/sepsis**.	**Red streaks = lymphangitis**
Chylous Effusions	Rupture of lymphatics due to **tumor obstruction → chylous ascites, chylothorax, chylopericardium**.	**Milky lymph in cavities**
Arteriosclerosis – 3 Types	1) **Arteriolosclerosis** 2) **Mönckeberg medial calcific sclerosis** 3) **Atherosclerosis**.	**Know the 3 patterns**

Arteriolosclerosis	Small arteries/arterioles; caused by **HTN & DM → wall thickening, luminal narrowing**, end-organ ischemia → chronic renal failure.	**HTN + DM → kidney ischemia**
Mönckeberg Sclerosis	**Calcification of tunica media**, age >50, **does NOT narrow lumen**, visible on X-ray.	**Media calcification without stenosis**
Atherosclerosis – Definition	Most important form; **intimal lesions (atheromas)** with **lipid core + fibrous cap** → luminal narrowing → ischemia.	**Intimal disease**
Atherosclerosis – Requirements	Requires **LDL deposition + inflammation**.	**LDL + chronic inflammation**
Key Cells in Plaque	**Macrophages**, **SMCs migrating from media**, **ECM production**, **extracellular lipid deposits**.	**SMC migration = hallmark**
Plaque Types	**Vulnerable**: thin cap, large lipid core, inflammation → rupture risk. **Stable**: thick cap, smaller core.	**Thin cap = rupture**
Major Risk Factors	**Age**, **male sex**, **smoking**, **hyperlipidemia**, **hypertension**, **diabetes**, **family history**.	**The Big 6**
Additional Risks	**Lp(a)**, **hyperhomocysteinemia**, **metabolic syndrome**, **CRP**, stress, obesity, inactivity, high-carb diet.	**Lp(a) & homocysteine**

Lecture 5 aneurysms and dissections

Topic	Key Concepts (Complete + Organized)	🔥 High-Yield Anchors (Bold)
Aneurysm – Definition	Localized **abnormal dilation** of an **artery or the heart**. Shapes: **Saccular** (portion of wall) vs **Fusiform** (circumferential).	**Localized dilation**, **Saccular**, **Fusiform**
True Aneurysm	All **three layers intact** (intima, media, adventitia). Examples: **atherosclerotic**, **syphilitic**, **congenital**, **ventricular aneurysm after MI**, **Berry aneurysms**.	**All 3 layers intact**, **Berry aneurysm**
Berry Aneurysm	Small **saccular** aneurysms at **Circle of Willis** due to **congenital medial weakness**; dilation is **permanent**.	**Circle of Willis**, **congenital wall defect**
False Aneurysm (Pseudo-aneurysm)	**Breach in vessel wall** → blood escapes but is contained by **extravascular connective tissue** → **pulsating hematoma**. Causes: **ventricular rupture after MI**, **vascular graft leak**.	**Pulsating hematoma**, **wall defect**, **not all layers**
True Aneurysm – Shapes	**Saccular**: spherical outpouching, may contain thrombus. **Fusiform**: long segment dilation, circumference expanded.	**Shape ≠ specific disease**

Aortic Aneurysm	Can involve **ascending, arch, descending, abdominal aorta**. Large aneurysms behave like a **mass** and may **rupture** or **compress** surrounding structures (e.g., **left recurrent laryngeal nerve → hoarseness**).	**High rupture risk**, **mass effect**
Major Causes of Aortic Aneurysm	**1) Atherosclerosis (most common)** → intimal plaques compress media → ischemia → **media thinning/weakening** → dilation. **2) Medial degeneration**: **HTN**, trauma, congenital (Berry), **Marfan**, **vasculitis**, **mycotic aneurysm**, immune injury.	**Atherosclerosis**, **HTN**, **Marfan**, **media degeneration**
AAA – Abdominal Aortic Aneurysm	Atherosclerotic aneurysm **most common in abdominal aorta**. Typically in **men**, rare < **50 years**. Often **below renal arteries and above bifurcation**.	**Men**, **below renal arteries**, **atherosclerosis**
AAA – Pathogenesis	Risk factors: **atherosclerosis**, **Marfan (fibrillin defect)**, **vasculitis**, **connective tissue degradation**, **inflammation**, **mycotic infection**.	**Fibrillin defect**, **proteolytic damage**, **mycotic**
AAA – Morphology	Size may reach **15 cm diameter**, **25 cm length**. Often **saccular or fusiform**, contains **laminated mural thrombus**, has **media thinning**.	**Large size = high rupture risk**, **mural thrombus**
AAA – Clinical Consequences	**Rupture** → catastrophic hemorrhage (**risk ↑ when ≥ 5 cm**). **Embolism** from mural thrombus. **Obstruction** of downstream vessels. **Compression** of ureters/vertebrae. **Pulsating abdominal mass**.	**≥5 cm rupture risk**, **pulsatile mass**, **mural emboli**
Mycotic Aneurysm	Vessel wall infection → **weakening + dilation**. Not limited to fungi. Causes: **septic emboli**, extension from **adjacent abscess**, **bacteremia seeding atherosclerotic plaque**.	**Infectious wall weakening**, **infective endocarditis source**
Syphilitic Aneurysm	**Tertiary syphilis** → immune-mediated **obliterative endarteritis of vasa vasorum** → **ischemic injury of media** → dilation of aorta and **aortic root**, → **aortic regurgitation**. Not a mycotic aneurysm.	**Vasa vasorum obliteration**, **media ischemia**, **aortic regurgitation**
Arterial Dissection – Definition	**Intimal tear** → blood enters media → **separates layers**, forms **false lumen**.	**Intimal tear**, **false lumen**
Aortic Dissection – Pathogenesis	Blood enters through tear → tracks through media under pressure. Causes: **HTN (major)**, **atherosclerosis**, **Marfan**, **Ehlers–Danlos**, **vitamin C deficiency**, **copper metabolism defects**.	**HTN = #1 risk**, **connective tissue defects**
Consequences of Dissection	**Rupture**, **massive hemorrhage**, **cardiac tamponade**, **weak distal pulses**, **limb ischemia**,	**Tamponade**, **rupture**, **limb ischemia**

	aortic regurgitation, **organ compression**, **mural thrombus**.	
Clinical Presentation of Dissection	**Sharp tearing chest/back pain**, **hypotension**, **shock**, **absent/weak pulses**, rapid progression to **death** if ruptured.	**Tearing pain radiating to back**
Dissection Classification – Type A	**Most common**, **more dangerous**. **Involves ascending aorta** (± arch ± descending). Requires urgent surgery.	**Type A = proximal**, **ascending involvement**
Dissection Classification – Type B	**Distal** to major branches; does **not involve ascending aorta**; begins **distal to subclavian**; managed medically unless complications arise.	**Type B = distal**, **DeBakey III**
Marfan Syndrome Role	**Autosomal dominant** mutation in **fibrillin** → **defective elastin synthesis** → weak media → **aneurysm + dissection**. Manifestations: **long limbs**, **lens subluxation**, **aortic disease**.	**Fibrillin defect**, **aortic dissection risk**
Imaging for Diagnosis	**CT angiography**, **MRI**, **Transesophageal echo**, **X-ray** (widened mediastinum).	**CT angiography = gold standard**
Prognosis	Improved with **rapid diagnosis**, **antihypertensives**, **surgery (graft, plication)**. Still **highly fatal** due to coexistence with CVD.	**Early intervention saves lives**

Lecutre 6 hypertensive vascular disease

Main Topic	Detailed Summary (Complete Content)	High-Yield Points (Bold)
Definition of BP & Tools	BP measured via **sphygmomanometer or digital monitor** → gives **systolic + diastolic pressures**.	**Systolic**, **Diastolic**
Types of Hypertension (Severity)	**Benign HTN (95%)** vs **Malignant HTN (5%)**.	**95% benign**, **5% malignant**
Types of Hypertension (Etiology)	**Primary (essential) HTN = 95%**, no identifiable cause. **Secondary HTN = 5%**, due to identifiable causes (e.g., **renal disease**, **renal artery stenosis**).	**Essential 95%**, **Secondary 5%**, **Renal disease = most common secondary cause**
Malignant (Accelerated) Hypertension	**Systolic > 200 mmHg**, **Diastolic > 120 mmHg**; rapidly progressive; fatal in **1–2 years** if untreated. Causes **renal failure**, **retinal hemorrhages**, **end-organ damage**. Usually develops on top of pre-existing benign HTN.	**>200/120**, **End-organ damage**, **Retinal hemorrhages**, **Renal failure**
Hypertension Complications	**Stroke**, **multi-infarct dementia**, **coronary atherosclerosis**, **LV hypertrophy → heart failure**, **aortic dissection**, **renal failure**, **retinal hemorrhages**.	**Stroke**, **LVH**, **Aortic dissection**, **Renal failure**

Essential HTN – Pathogenesis	**Multifactorial**: 1) **Genetic factors** (familial clustering, **angiotensinogen polymorphisms**, **Ang II receptor variants**, genes regulating **renal Na^+ absorption**). 2) **Environmental**: **stress**, **obesity**, **smoking**, **physical inactivity**, **high salt intake**.	**Genetic + environmental**, **RAAS polymorphisms**, **Na^+ absorption genes**, **Salt intake**
Blood Vessels in HTN – General Morphology	HTN damages **small arteries & arterioles → arteriolosclerosis**. Two subtypes: **Hyaline arteriolosclerosis** and **Hyperplastic arteriolosclerosis**.	**Arteriolosclerosis**, **Small arteries**
Hyaline Arteriolosclerosis	Associated with **benign HTN**. Mechanism: **plasma protein leakage** across **injured endothelium** + **ECM overproduction by smooth muscle**. Morphology: **homogeneous pink hyaline thickening**, **luminal narrowing**.	**Benign HTN**, **Protein leakage**, **ECM deposition**, **Luminal narrowing**
Hyaline Arteriolosclerosis – Complications	Can affect any organ, but most severe in **kidneys → nephrosclerosis → chronic renal failure**. Other causes even without HTN: **diabetes**, **elderly age**.	**Nephrosclerosis**, **Chronic renal failure**, **Diabetes**
Hyperplastic Arteriolosclerosis	Occurs in **malignant HTN**. Morphology: **Onion-skin concentric laminated thickening**, **redundant/reduplicated basement membranes**, **luminal narrowing**, **fibrinoid necrosis (necrotizing arteriolitis)**.	**Malignant HTN**, **Onion-skin**, **Fibrinoid necrosis**, **Basement membrane duplication**
Onion-Skin Appearance Explanation	Due to **multiple duplicated layers of basement membrane**, giving appearance of sliced onion rings; causes **severe luminal narrowing**.	**Basement membrane duplication**, **Severe narrowing**
Necrotizing Arteriolitis	Seen in **malignant HTN**. **Fibrinoid necrosis** of arteriolar wall; leads to acute ischemia.	**Fibrinoid necrosis**, **Malignant HTN**
End-Organ Damage in HTN	The organs most susceptible: **Heart**, **Brain**, **Kidneys**, **Aorta**, **Retina**. Mechanisms: **arteriolosclerosis**, **infarction**, **hemorrhage**, **ischemia**, **accelerated atherosclerosis**.	**Target-organ damage**, **Heart–Brain–Kidney–Aorta–Retina**
Summary of HTN Effects on Organs	**Heart**: LVH → HF. **Brain**: stroke, dementia. **Kidneys**: nephrosclerosis → renal failure. **Eyes**: retinal hemorrhage. **Aorta**: dissection risk ↑.	**LVH**, **Stroke**, **Renal failure**, **Retinal damage**, **Aortic dissection**

Lecture 7 ischemic heart disease

Main Topic	Complete Detailed Summary	High-Yield Anchors (Bold)
Definition of IHD	Group of syndromes caused by **myocardial ischemia** → imbalance between **blood supply** & **oxygen demand**. IHD ≈ **Coronary artery disease (CAD)**.	**Ischemia = supply < demand, CAD = 90% atherosclerosis**
Mechanisms of Reduced Supply	**Atherosclerosis (90%)**, **thrombosis**, **vasospasm**, **stenosis**, **shock**, **hypovolemia**.	**Atherosclerosis = #1 cause**
Mechanisms of Increased Demand	**Tachycardia**, **exertion**, **hypertension**, **stress**, **fever**, ↑ contractility.	**↑ HR = ↑ demand**
Mechanisms of ↓ O_2-Carrying Capacity	**Severe anemia**, **CO poisoning** (CO competes with O_2 for Hb). Least common cause.	**CO poisoning, severe anemia**
Main Clinical Syndromes of IHD	1) **Angina pectoris** (ischemia but **no necrosis**). 2) **Acute MI** (ischemia **with necrosis**). 3) **Chronic IHD → heart failure**. 4) **Sudden cardiac death (SCD)**.	**Angina, MI, Chronic IHD, SCD**
Angina Pectoris – Definition	**Intermittent chest pain** due to **transient reversible ischemia** (<20 min), **no myocyte death**. Pain radiates to **left arm, jaw, neck, epigastrium**.	**Pain <20 min, relieved by rest/nitroglycerin**
Stable (Typical) Angina	Triggered by **exertion**; due to **critical stenosis >75%**; relieved by rest or **sublingual nitroglycerin**.	**Critical stenosis, exertional, relieved by rest**
Prinzmetal (Variant) Angina	Occurs **at rest**, due to **coronary vasospasm**, often in arteries without atherosclerosis. Treat with **vasodilators (nitroglycerin, CCBs)**.	**Rest pain, vasospasm, CCB responsive**
Unstable Angina (Crescendo)	Increasing frequency/intensity of pain, **at rest**, lasts longer. Cause: **plaque rupture**, **partial thrombosis**, **distal embolization**, **vasospasm**. **Pre-infarction angina** → one step before MI.	**Pre-MI, plaque rupture, partial thrombus, rest pain**
Myocardial Infarction (MI)	**Necrosis** of myocardium due to prolonged ischemia. Most commonly due to **acute plaque rupture + thrombosis**.	**Necrosis, coronary thrombosis, LAD = 40–50%**
MI Clinical Manifestations	**Severe crushing chest pain** radiating to **left arm/jaw**; **not relieved by rest/nitroglycerin**; **weak rapid pulse**, **dyspnea**, **sweating**, possible **cardiogenic shock** if >40% LV infarct.	**Pain >20 min, not relieved, dyspnea, shock**

Silent MI	MI **without symptoms**; seen in **diabetics**, **elderly**, **ICU patients**. Diagnosed by **ECG + biomarkers**.	**Diabetics**, **elderly**, **silent infarct**
MI Causes	**Acute thrombosis over ruptured plaque**. **LAD occlusion = 40–50%** of MIs.	**LAD = widow maker**
Diagnosis of MI	1. **Symptoms** 2. **ECG changes** 3. **Biomarkers** leaking from necrotic myocardium.	**Symptoms + ECG + biomarkers**
Cardiac Biomarkers	**Troponins T & I** = **best markers**. Rise 2–4 h, peak 24–48 h, remain elevated 7–10 days. **CK-MB** = 2nd best, helpful for detecting **reinfarction**. Others: **myoglobin**, **LDH**.	**Troponin = gold standard**, **CK-MB for reinfarction**
MI Histologic Evolution		
0–24 hours	**Coagulative necrosis**, **wavy fibers**, edema.	**Coagulative necrosis**
1–3 days	**Neutrophil infiltration** (acute inflammation).	**Neutrophils**
3–7 days	**Macrophages** remove dead cells → risk of **rupture** highest.	**Macrophages**, **rupture window**
7–14 days	**Granulation tissue** (loose CT + new capillaries).	**Granulation tissue**
Weeks → Months	**Dense collagenous scar** forms; permanent loss of function.	**Scar**, **no regeneration**
Major MI Complications		
1. Death	50% die before reaching hospital (usually **ventricular fibrillation → SCD**).	**VF = most common cause of early death**
2. Cardiogenic Shock	Occurs when **>40% LV infarct**; mortality **70%**.	**Shock = worst in-hospital complication**
3. Myocardial Rupture	3 types: **free wall → tamponade**, **septal → VSD**, **papillary muscle → severe MR**.	**Tamponade**, **VSD**, **MR**
4. Pericarditis	Occurs **2–3 days post-MI**, immune-mediated; resolves.	**Fibrinous pericarditis**
5. Infarct Expansion	Stretching/dilation of infarct zone, especially **anteroseptal MIs**.	**Expansion = thinning/dilation**
6. Mural Thrombus	Due to **stasis + endocardial injury** → embolization risk.	**Thromboembolism**
7. Ventricular Aneurysm	**Late complication**, usually from **large transmural anteroseptal MI**; wall becomes thin scar → **true aneurysm**. Complications: **mural thrombus, arrhythmias, HF**.	**True aneurysm**, **thin scar**, **arrhythmias**
8. Progressive Heart Failure	Progressive decline due to loss of myocardium + compensatory hypertrophy exhaustion.	**Chronic IHD → HF**

Sudden Cardiac Death (SCD)	Unexpected death within **<1 hour** of symptoms; due to **lethal arrhythmia (ventricular fibrillation)**. Most common underlying cause: **CAD (atherosclerosis)**. In young: **HCM**, **myocarditis**, **valve disease**, **congenital coronary anomalies**, **conduction defects**.	**VF = mechanism, CAD = cause, young = non-atherosclerotic causes**

Lecture 8 valvular heart disease

Category	Complete High-Yield Summary	High-yield
Basic Concepts	Valvular disease = **stenosis** (failure to open) OR **regurgitation** (failure to close). Can involve **cusps**, **annulus**, **chordae tendineae**, **papillary muscles**. Can be **acute** (e.g., chordal rupture after MI) or **chronic** (calcification, scarring).	**Stenosis = narrow; Regurgitation = backflow**
Stenosis vs. Regurgitation	**Stenosis** → chronic process, calcification, post-inflammatory scarring → obstructed forward flow. **Regurgitation** → abnormal closure → backward flow → volume overload.	**Stenosis = chronic; Regurg = acute/chronic**
Clinical Clues	**Murmurs**; **thrills** (palpable murmur); signs depend on valve involved.	**Murmur = hallmark**
Congenital Valve Disease	Most common = **bicuspid aortic valve** (2 cusps instead of 3). Seen in 1–2%. Often silent early. Later → **early degenerative calcification**, **aortic stenosis**, **LV hypertrophy**, **HF**.	**Bicuspid = most common congenital lesion**
Why Bicuspid Valve Calcifies Early	Abnormal cusp geometry → ↑ mechanical stress → accelerated wear and calcification → premature stenosis.	**↑ Stress → ↑ Calcification**
Acquired Valve Disease – Overview	Mitral valve = most commonly affected (left-sided valves exposed to ↑ pressure & shear stress). Most common cause worldwide = **rheumatic fever**. Other cause = **infective endocarditis (IE)**.	**Mitral = most affected; RF = #1 worldwide cause**
Rheumatic Fever (RF) – Etiology	**Immune-mediated**, not an infection. Due to **molecular mimicry**: anti–Group A Streptococcus antibodies cross-react with **heart, joints, skin, brain**. Occurs after **pharyngitis** or **skin infection**.	**Molecular mimicry, post-strep immune reaction**
RF Epidemiology	Common in **children** (pharyngitis), especially where penicillin access is limited.	**Children, low-resource settings**
RF Pathogenesis	Type II hypersensitivity; antibodies against **streptolysin O**, **DNAse B**. Time gap: symptoms appear **weeks after infection**.	**Anti-strep antibodies**
RF – Acute Phase	Occurs in **80% children**. Manifestations: **fever**, **migratory polyarthritis**, **carditis** (can → arrhythmias, myocarditis, chamber dilation,	**JONES major criteria**

	functional MR), **Sydenham chorea**, **skin lesions (erythema marginatum, subcutaneous nodules)**. Labs: ↑ anti-streptolysin O.	
Carditis in RF – Acute Morphology	**Aschoff bodies** = pathognomonic: foci of **T-cells, plasma cells, activated macrophages (Anitschkow cells)**. **Valve vegetations** = small, sterile verrucae.	**Aschoff = diagnostic**
RF – Diagnosis	**JONES criteria** (not required in detail). Blood cultures usually **negative** (infection resolved).	**Major criteria = JONES**
RF – Chronic Phase	Occurs **years to decades later**. Chronic inflammation → **scarring + calcification**. Functional result = **stenosis > regurgitation**. **Mitral valve most commonly affected** → LA dilation → AF → mural thrombi → emboli.	**Chronic RF = mitral stenosis**
RF – Chronic Morphology	Thickened, fused cusps; commissural fusion; **fish-mouth or buttonhole stenosis**; shortened chordae; calcifications. Aschoff bodies rare in chronic stage.	**Fish-mouth stenosis**
Progression in Chronic RF	Chronic scarring → **hemodynamic obstruction**, **murmurs**, **CHF**, **arrhythmias**, **thromboembolism**.	**Stenosis → LA dilation → AF**
Infective Endocarditis (IE)	True **microbial infection** of valves/endocardium. Produces **friable, destructive vegetations** containing **organisms + thrombus + necrotic debris**. Causes **septic emboli** and systemic complications.	**Vegetations with organisms**
IE Predisposing Factors	Congenital disease, chronic RF, prosthetic valves, catheters, immunodeficiency, IV drug use, septicemia. Dental procedures in high-risk patients can trigger IE.	**IV drug use → tricuspid IE**
IE Classification	**Acute IE** → highly virulent (e.g., Staph aureus), attacks normal valves, rapid onset, high mortality. **Subacute IE** → low virulence (Strep viridans), infects previously abnormal valves, slow onset, better prognosis.	**Acute = S. aureus**, **Subacute = S. viridans**
IE Clinical Features	**Fever**, **chills**, **new murmur**, **embolic events**, splinter hemorrhages, Osler nodes, Janeway lesions.	**Fever + new murmur**
IE Diagnosis	**Blood cultures + Echocardiography** (vegetation). Duke criteria exist but not needed.	**Blood culture = essential**
IE Complications	**Septic emboli**, **abscesses**, **septic infarcts**, **mycotic aneurysms**, valve destruction → **acute regurgitation**, HF, death.	**Septic emboli**, **abscess**, **mycotic aneurysm**
IE Morphology	Vegetations = **large, friable, destructive**. Most common on **mitral and aortic valves**; **tricuspid valve** in IV drug users.	**Bulky vegetations**
Management of IE	Long-term (≥6 weeks) **IV antibiotics**; valve replacement if needed.	**IV therapy required**

Lecture 9 cvs tumors

Category	Full High-Yield Description	Key Identifiers
Definition: Vascular Tumors	Diverse neoplasms derived from **endothelial cells**. Classified by biological behavior into **benign, borderline (intermediate), and malignant**.	**Endothelial origin**
Benign Vascular Tumors	• Contain **well-formed vascular channels** resembling normal vessels.• Lined by **flat, normal-looking endothelial cells** (minimal/no atypia).• **No metastasis**.• Most common example = **Hemangioma**.	**Organized vessels, bland cells**
Borderline / Intermediate Tumors	• Behavior **between benign and malignant**.• **More cellular**, **cytologic atypia**, increased proliferation.• Partly form abnormal channels; sometimes spindle-shaped cells.• Locally aggressive but **rare metastasis**.Example = **Kaposi Sarcoma**.	**Atypia + limited aggression**
Malignant Vascular Tumors	• **Do NOT form well-organized vessels**.• **Marked atypia**, pleomorphism, mitoses, anaplasia.• Infiltrative, destructive growth + metastasis.Example = **Angiosarcoma**.	**Disorganized, aggressive, metastatic**
Important Nomenclature	• **–oma** = generally benign.• **Sarcoma** = malignant mesenchymal tumor.• **Hem-angi-oma** = tumor of **blood vessels**.• **Lymph-angi-oma** = tumor of **lymphatic channels** (no blood).• **Angiosarcoma** = malignant endothelial tumor.	**Prefix indicates vessel type**
Hemangioma — General	• **Most common benign vascular tumor**.• Composed of **blood-filled vessels**.• **Most common age group: infants & children**.• Often present at birth.• **Many regress spontaneously**.• Most common location: **head and neck**.• 1/3 internal → especially **liver**.• Malignant transformation: **very rare**.	**Infancy, spontaneous regression**
Capillary Hemangioma	• Most common variant.• Affects **skin, oral mucosa, lips**.• Histology: **capillary-sized vascular spaces**.• Gross: **strawberry red** or bruise-like.	**Capillary-like spaces**
Strawberry (Juvenile) Hemangioma	• Seen in newborns, especially **head & neck**.• Appears at birth → **regresses over months/years**.• Excellent prognosis.	**Birth + regression**
Pyogenic Granuloma	• Rapid-growing **pedunculated** lesion on **gingiva**.• 1/3 have history of trauma.• Misnomer: **not pyogenic, not granuloma**.	**Gingival, trauma-related**
	•	

Cavernous Hemangioma	Large, **dilated vascular spaces** ("caverns").• Common in **deep organs**, especially **liver**.• **Does NOT regress** spontaneously.• More clinically significant due to location.	**Deep organs, large channels**
Kaposi Sarcoma — Overview (Borderline)	• Vascular neoplasm caused by **HHV-8**.• Requires **immunosuppression** to develop.• 4 types: **Classic, Endemic (African), Transplant-associated, AIDS-associated**.• **AIDS-associated = epidemic KS**, the **most common HIV-related malignancy** → AIDS-defining.	**HHV-8 + immunosuppression**
Kaposi Sarcoma — Pathogenesis	• **HIV** → ↓ T-cell immunity.• **HHV-8** reactivation & cytokines → endothelial proliferation → tumor.	**HIV + HHV-8 synergy**
Kaposi Sarcoma — Clinical & Histology	• Red-purple **skin plaques and nodules**, typically **distal lower limbs**, spreading proximally.• Histology: **Spindle-shaped endothelial cells**, crowded; poor formation of vascular channels.	**Spindle cells, purple lesions**
Angiosarcoma — Overview (Malignant)	• Highly malignant endothelial tumor.• Sites: **skin, soft tissue, breast, liver**.• Aggressive, infiltrative, poor prognosis.	**Aggressive endothelial malignancy**
Angiosarcoma — Risk Factors	1. **Chemical carcinogens** (especially liver angiosarcoma): • **Vinyl chloride**, **arsenic**, **Thorotrast**.2. **Radiation exposure** (post-radiotherapy).3. **Chronic lymphedema** (e.g., after mastectomy → upper limb swelling → Stewart–Treves syndrome).4. **Long-term foreign bodies** (rare).	**Vinyl chloride → liver**
Cardiac Tumors — Overview	• Very rare, but **location is critical**.• **Metastatic tumors are more common than primary**.• ~5% of cancer deaths show cardiac metastasis.• Most common primary malignant = **Angiosarcoma**.• Benign tumors exist but are clinically important due to obstruction or embolization.	**Metastases > primary tumors**
Most Common Metastatic Source to Heart	**Lung cancer**.	**Lung → heart**
Clinical Significance of Cardiac Tumors	Depends on **location**, **mobility**, **size**, **friability**, **patient age**, and **tumor biology**.	**Location determines symptoms**

Cardiac Tumors — Key Features	**Ball-valve effect:** Pedunculated tumor moves with blood flow → intermittent obstruction of a valve.**Embolization:** Fragile pieces break → systemic emboli.**Fever & malaise:** Tumor releases IL-6 → inflammatory symptoms.**Diagnosis: Echocardiography**, CMRI.**Treatment:** Surgical excision for benign tumors; depends on type/location for others.	**Stalk = ball-valve obstruction**